on STN
ACCESSION NUMBER: 92371258 EMBASE
DOCUMENT NUMBER: 1992371258
TITLE: Maitotoxin induces a calcium-dependent membrane depolarization in GH4C1 pituitary cells via activation of type L voltage-dependent calcium channels.
AUTHOR: Xi D.; Van Dolah F.M.; Ramsdell J.S.
CORPORATE SOURCE: Marine Biomedical/Environmental Sci., Medical University of South Carolina, 221 Fort Johnson Rd.,Charleston, SC 29412, United States
SOURCE: Journal of Biological Chemistry, (1992) 267/35 (25025-25031).
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB . . . water-soluble polyether, isolated from the marine dinoflagellate Gambierdiscus toxicus, that stimulates hormone release and Ca2+ influx. We have investigated the **action** by which MTX induces Ca2+ influx and stimulates prolactin (PRL) release from GH4C1 rat pituitary cells. PRL release elicited by MTX is abolished in a concentration-dependent manner by nimodipine, a dihydropyridine (DHP) antagonist of type L **voltage**- dependent calcium channels (L-VDCC), indicating that MTX-enhanced PRL release occurs via activation of type L-VDCC. As an initial approach to. . . site. The effect of MTX on DHP binding was largely (65%) calcium-dependent. We next examined whether MTX alters the membrane **potential** of GH4C1 cells using the **potential sensitive** fluorescent **dye** bisoxonol. Addition of 100 ng/ml MTX to GH4C1 cells caused a membrane depolarization within 2.5 min which reached a plateau. . . MTX-induced depolarization was not prevented by substitution of impermeant choline ions for Na+. It was similarly unaffected by K+ channel **blockers** or by depleting the K+ chemical concentration gradient with gramicidin, a monovalent cation pore-forming agent. By contrast, low extracellular Ca2+. . . with a component of the VDCC complex, which, in turn, initiates a positive feedback mechanism involving calcium-dependent membrane depolarization and **voltage**-dependent activation of calcium channels.

L4 ANSWER 15 OF 19 EMBASE
on STN
ACCESSION NUMBER: 92240513 EMBASE
DOCUMENT NUMBER: 1992240513
TITLE: Maitotoxin-induced intracellular calcium rise in PC12 cells: Involvement of dihydropyridine-sensitive and ω-conotoxin-sensitive calcium channels and phosphoinositide breakdown.
AUTHOR: Meucci O.; Grimaldi M.; Scorziello A.; Govoni S.; Bergamaschi S.; Yasumoto T.; Schettini G.
CORPORATE SOURCE: Section of Pharmacology, Human Communicative Sciences Dept., II School of Medicine, Via S. Pansini 5,80131 Napoli, Italy
SOURCE: Journal of Neurochemistry, (1992) 59/2 (679-688).
ISSN: 0022-3042 CODEN: JONRA
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
052 Toxicology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB . . . calcium concentration and are always associated with an increase of the free cytosolic calcium level. We tested the effects of **voltage-sensitive** calcium channel **blockers**

(nicardipine and ω-conotoxin) on maitotoxin-induced intracellular calcium increase, membrane depolarization, and inositol phosphate production in PC12 cells. Maitotoxin dose dependently. . . was reduced by pertussis toxin pretreatment. Maitotoxin caused a substantial membrane depolarization of PC12 cells as assessed by the fluorescent **dye** bisoxonol. This effect was reduced by pretreating the cells with either nicardipine or ω-conotoxin and was almost completely abolished by. . . in a calcium-free EGTA-containing medium. The findings on maitotoxin-induced cytosolic calcium rise and membrane depolarization suggest that maitotoxin exerts its **action** primarily through the activation of **voltage-sensitive** calcium channels, the increase of inositol phosphate production likely being an effect dependent on calcium influx. The ability of nicardipine and ω-conotoxin to inhibit the effect of maitotoxin on both calcium homeostasis and membrane **potential** suggests that L- and N-type calcium channel activation is responsible for the influx of calcium following exposure to maitotoxin, and. . .

L4 ANSWER 16 OF 19 EMBASE
on STN

ACCESSION NUMBER: 92249069 EMBASE
DOCUMENT NUMBER: 1992249069
TITLE: Membrane properties of identified mesencephalic dopamine neurons in primary dissociated cell culture.
AUTHOR: Chiodo L.A.; Kapatos G.
CORPORATE SOURCE: 1261 Scott Hall, Wayne State Univ. School of Medicine, 540 E. Canfield Ave.,Detroit, MI 48201, United States
SOURCE: Synapse, (1992) 11/4 (294-309).
ISSN: 0887-4476 CODEN: SYNAET
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 001 Anatomy, Anthropology, Embryology and Histology
002 Physiology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB . . . their distinct morphology, and this identification was validated with a double-labeling procedure that entailed the intracellular deposition of a fluorescent **dye** (Lucifer yellow or ethidium bromide), followed by processing for tyrosine hydroxylase immunocytochemistry. DA neurons identified in this manner were observed to have resting membrane **potentials** between -50 and -75 mV, input resistances of 50-360 MΩ, and membrane time constants of 4.1-14.1 msec. Forty-seven percent of. . . cells displayed spontaneous activity that was irregular in nature and often contained bursts (burst length was between two and six **action potentials**). The DA neurons displayed a variety of ionic conductances, including (1) a Na+ conductance (g(Na)) that underlies the **action potential**, (2) Ca2+ conductances (g(Ca)) that mediate the nonsomatic low- and high-threshold spikes observed, and (3) at least three K+ conductances (g(K)). **Voltage**-clamp analysis revealed several distinct transmembrane ionic currents, including (1) a large, rapidly inactivating tetrodotoxin-**sensitive** inward Na+ current (I(Na)), (2) a 4-aminopyridine-**sensitive**, transient early outward K+ current that required a conditioning hyperpolarization of the membrane to be activated by a subsequent depolarization. . . current was Ca2+-dependent and was not affected by tetraethylammonium ions. This current was termed I(AHP). The remaining current was not **sensitive** to changes in the extracellular Ca2+ concentration but was blocked by external tetraethylammonium. This current was termed I(K). The direct. . . (1-200 μM) onto the soma dose-dependently hyperpolarized these neurons; this effect was potentiated by the presence of the catecholamine reuptake **blocker** cocaine hydrochloride (10-200 μM). Under **voltage**-clamp conditions, DA was observed to increase I(K) significantly and had little effect on I(AHP). Thus, DA neurons in

monolayer cultures. . .

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ACCESSION NUMBER: 91049471 EMBASE
DOCUMENT NUMBER: 1991049471
TITLE: Bretylium causes a K+-Na+ pump activation that is independent of Na+/H+ exchange in depolarized rat, mouse and human lymphocytes.
AUTHOR: Tron L.; Pieri C.; Marian T.; Balkay L.; Emri M.; Damjanovich S.
CORPORATE SOURCE: Biomedical Cyclotron Laboratory, University Medical School of Debrecen, Debrecen, Hungary
SOURCE: Molecular Immunology, (1990) 27/12 (1307-1311). ISSN: 0161-5890 CODEN: IMCHAZ
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB We have studied a bretylium tosylate induced increase of the membrane **potentials** of partially depolarized rat, mouse and human lymphocytes, using the **potential sensitive dye** , bis [1,3 dibutyl-barbituric acid-(5)- trimethine oxonol]. The extent of this depolarization is dose-dependent and decreased in magnitude as the temp was reduced from 37°C to room temperature The repolarizing effect is inhibited by K+-Na+-pump **blockers** or lack of extracellular Na+. Sodium ion channel **blockers** are effective in abolishing repolarization only if applied prior to, or simultaneously with, bretylium. Activation of Na+/H+ exchange is not. . . . is completely eliminated in the presence of 10 μM amiloride (concn of the diuretics having no measurable inhibition on the **action** of the exchanger). These data suggest that bretylium opens ligand- and **voltage** -gated Na+ channels, and repolarization occurs due to higher activity of the K+-Na+-pump stimulated by the enhanced intracellular Na+ accumulation.

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ACCESSION NUMBER: 87120609 EMBASE
DOCUMENT NUMBER: 1987120609
TITLE: Maps of optical action potentials and NADH fluorescence in intact working hearts.
AUTHOR: Salama G.; Lombardi R.; Elson J.
CORPORATE SOURCE: Department of Physiology, University of Pittburgh School of Medicine, Pittsburgh, PA 15261, United States
SOURCE: American Journal of Physiology - Heart and Circulatory Physiology, (1987) 252/2 (21/2) (H384-H394). CODEN: AJPPDI
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 002 Physiology
018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index
LANGUAGE: English
AB **Voltage-sensitive dyes** were used to stain intact perfused hearts and to simultaneously measure optical **action potentials** (APs) from 124 sites on the epicardium. Patterns of electrical depolarization (activation) and repolarization (recovery) along the surface of the. . . . be altered by electrical stimulation. The normal heterogeneities in AP durations became more pronounced in the presence of the Ca2+-entry **blocker**, verapamil. The local metabolic state of the tissue was also monitored optically through its intrinsic NADH fluorescence measured from 124. . .

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ACCESSION NUMBER: 80026362 EMBASE
DOCUMENT NUMBER: 1980026362
TITLE: The effects of some organic 'calcium antagonists' on calcium influx in presynaptic nerve terminals.
AUTHOR: Nachshen D.A.; Blaustein M.P.
CORPORATE SOURCE: Dept. Physiol. Biophys., Washington Univ. Med. Sch., St Louis, Mo. 63110, United States
SOURCE: Molecular Pharmacology, (1979) 16/2 (579-586).
CODEN: MOPMA3
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
030 Pharmacology
002 Physiology
008 Neurology and Neurosurgery
LANGUAGE: English

AB The **actions** of the organic 'Ca antagonists' verapamil and D-600 were tested on pinched-off presynaptic nerve terminals (synaptosomes) from rat brain, and. . . or veratridine, an alkaloid that opens sodium channels. The extra uptake induced by depolarizing media appears to be mediated by **voltage-sensitive** Ca channels. Synaptosome depolarization was indirectly determined with the **voltage-sensitive** fluorescent **dye**, di-pentyl oxacarbocyanine. Verapamil or D-600 (100 μM) inhibited the K+-induced 45Ca uptake by about two thirds, but had no effect. . . observations indicate that Na channels as well as Ca channels are inhibited by verapamil and D-600. Recordings of miniature end-plate **potentials** were used to evaluate the **actions** of verapamil and D-600 at the frog neuromuscular junction, after miniature end-plate **potential** frequency had been made **sensitive** to changes in the bathing Ca concentration by raising the external K+. Miniature end-plate **potential** frequency was not affected by verapamil (40-50 μM) or D-600 (10 μM) but was significantly reduced by Mn2+ (0.2 mM), a known **blocker** of Ca channels. Although verapamil and D-600 appear to be very potent antagonists of Ca currents in heart and smooth muscle, we conclude that Ca channels in vertebrate neurons are much less **sensitive** to these drugs.

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